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Human Serum from Urban and Rural Adolescents and Their Mothers Shows Exposure to Polychlorinated Biphenyls Not Found in Commercial Mixtures

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Abstract

Although polychlorinated biphenyls are no longer sold as commercial mixtures, they are still being produced through modern manufacturing processes. We have previously shown that non-Aroclor PCB11 is prevalent in indoor and outdoor air, sediment, and detected in human serum. Here we report the prevalence of non-Aroclor PCB congeners (≤0.20 weight percent in Aroclor) in human serum collected from urban and rural adolescents and their mothers. We hypothesized that additional non-Aroclors congeners are present in serum. Sera were extracted and detected for 209 PCBs using gas chromatography-tandem mass spectrometry. A list of 70 non-Aroclor PCB congeners was determined by measurement of original Aroclors. PCB 11, 14, 35 and 209 are the major dominating and most frequently detected congeners. PCB 14 and 35 have not been previously reported for environmental matrices. Adolescents have significantly lower total non-Aroclor PCBs concentration than mothers in East Chicago ($p<0.001$) and Columbus Junction ($p=0.008$). There are significant differences in non-Aroclor PCBs between East Chicago community and Columbus Junction community ($p<0.001$). Non-Aroclor PCBs represent an average of 10% (and up to 50%) of total PCBs measured in serum. An average of 50% (and up to 100%) of these concentrations may attribute to aryl azo and phthalocyanine paint pigments.

Graphical abstract

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Supporting Information

Figures of comparison of lab measured SRM to NIST measured SRM 1957 and example chromatograms of samples analyzed using SPB-Octyl column and DB-5 column; tables of a list of ^{13}C labeled PCB surrogate standards and internal standards and their recovery, weight percent of 209 PCB congeners of our lab measured Aroclor, non-Aroclor PCB congener LOQ values. This material is available free of charge via Internet http://pubs.acs.org.

INTRODUCTION

Polychlorinated biphenyls (PCBs) are legacy organic compounds consisting of 209 congeners with different chemical structures. They are semi-volatile, persistent and bioaccumulating toxic pollutants. Beginning in late 1929, the Monsanto Chemical Company manufactured PCBs as Aroclor mixtures in United States for use in hydraulics fluids, plasticizers, flame retardants, electrical transformers and others.[1] In 2013, the International Agency for Research on Cancer (IARC) reclassified PCBs from Group 2A (probably carcinogenic) to Group 1 (carcinogenic to humans).[2] PCBs have also been identified as human endocrine disruptors[3, 4] and neurotoxicants.[3, 5–7]

PCBs are still detectable in many environmental matrices although their commercial production was banned in the late 1970s. Aroclor PCB congeners, as well as the mixtures themselves, have been well-studied in the environment[8–11] and in human specimens.[12–15] Toxicity studies on Aroclor PCB congeners such as PCB 153 have also been widely investigated. Non-Aroclor PCB congeners have not been a focus due to the assumption of their absence in the environment. Historically, analytical methods were developed using Aroclors as calibration standards. This approach specifically excluded non-Aroclor PCB congeners from being identified. Thus, there remains a paucity of data on non-Aroclor PCBs in the environment and on the potential for human exposure. PCB 11 was not produced in Aroclor mixtures and is widely measured in the environment[8, 16–20], organic paint pigments[21, 22] and consumer goods.[23] Recently, our group also discovered PCB 11 in human serum.[13, 14] Furthermore, another congener that was not present in commonly used Aroclor mixtures, PCB 209, is also detected in many matrices including human sera.[14, 15, 24–27] With detection of these PCBs in the environment, we hypothesize that additional non-Aroclor PCB congeners are present in human serum.

The main objective of this paper is to examine the prevalence of non-Aroclor PCB congeners in human serum collected from adolescents and their mothers living in East Chicago, Indiana and Columbus Junction, Iowa between April 2010 to March 2011 as part of the Airborne Exposure to Semi-volatile Organic Pollutants (AESOP) Study. Specifically, consistent with the Aroclors, we hypothesize that adolescents have lower non-Aroclor PCB

concentrations than mothers especially for the higher-chlorinated congeners. We also determined a list of PCB congeners as non-Aroclor PCBs. The presence of non-Aroclor PCBs in human serum indicates the importance for further studies to assess their potential toxicity and health risks to the general population and children in particular. Majority of these non-Aroclor congeners have never been examined and to our knowledge, no toxicity information is available. This may also contribute significance advances to future human health risk assessment.

MATERIALS AND METHODS

Study locations, participants & sample collection

Detailed information on study locations, participants and sample collection were presented in our previous publications.[13, 14, 17] Briefly, 183 blood samples were collected from school adolescents (ages 13–18 years) and their mothers (ages 29–58 years) living in East Chicago, Indiana (42 adolescents and 39 mothers) and Columbus Junction, Iowa (55 adolescents and 47 mothers) from April 2010 to March 2011. This is the first time we have reported data from these serum samples, although we have previously reported findings regarding PCBs from serum collected from most of the same participants in 2008 and 2009. Four households were excluded from this analysis due to instances of poor surrogate standards recoveries (2 East Chicago and 2 Columbus Junction mother-child pairs). The prevalence of non-Arcoclor PCBs serum data from the remaining 40 adolescents and 37 mothers from East Chicago and 53 adolescents and 45 mothers from Columbus Junction are reported in this study (N=175). Cohort demographic data are presented in Table 1. All protocols were reviewed and approved by University of Iowa Institutional Review Board. Written consent and assent were obtained in English or in Spanish from all participants.

Chemicals

Pesticide-grade solvents were used in this study. All 209 calibration standards were purchased from AccuStandard (New Haven, CT, USA). ^{13}C-labelled PCB mixtures were purchased as surrogate and internal standards from Cambridge Isotope Laboratories, Inc. (Andover, MA, USA). A list of ^{13}C-labelled PCB mixtures is included in Supporting Information (SI), Table S1. Standard reference material (SRM) 1957 (Organic Contaminants in Non-Fortified Human Serum) was purchased from National Institute of Standards and Technology (NIST), Gaithersburg, MD, USA. Aroclors 1016, 1221, 1242 and 1254 were obtained from Dr. Larry Robertson while Aroclor 1248 was purchased from AccuStandard.

Analytical & Instrument

PCBs in human serum were extracted using a method described in previous studies.[13, 14, 28] PCBs were analyzed and quantified using modified US EPA method 1668 in Agilent 7000 gas chromatography-tandem mass spectrometry (GC-MS/MS) under multiple reaction monitoring mode (MRM).[13, 14] Aroclor mixtures were diluted and analyzed in GC-MS/MS directly without involving in any extraction process. Two hundred and nine PCB congeners were quantified as single or co-eluting PCBs in 174 chromatographic peaks. A list of precursor-product transition can be found in our previous studies supporting information.[13, 14] PCB 11, PCB 14 and PCB 35 were further identified and verified using a

DB-5 column (30 m, 250 μm i.d. and 1.0 μm film thickness) in GC-MS/MS. An example chromatograms of PCB 11, 14 and 35 in samples and PCB standards using SPB-Octyl fused silica capillary column and DB-5 column was presented in SI, Figure S1.

Quality control

To evaluate our data quality, quality control samples (method blanks, surrogate standards and SRM) were included in the extraction and analysis steps of each batch. Non-Aroclor PCB congeners in the method blanks averaged 0.001 ± 0.008 ng.^{13}C-labelled PCB surrogate standards recoveries averaged 77% ± 19% (SI, Table S1). No significant difference was observed between each NIST SRM 1957 certified PCB congener value and our lab measured values (p=0.702) (SI, Figure S2). LOQ was determined on sum of the method blank mean plus twice the standard deviation (95% confidence interval). A value of zero (0) is assigned to each congener mass if the detected mass is below the limit of quantification (LOQ) as previously described.[13] A list of the LOQ values for the non-Aroclor PCB congeners can be found in SI, Table S2.

Statistical Analysis

All statistical analyses were performed using SPSS (IBM, Inc., Armonk, NY). Paired t-test was used to compare the weight percent of PCB congeners in original Aroclor measured in our laboratory and weight percent of PCB congeners in Aroclor reported by Frame *et al*.[29] The distribution of human serum concentrations were not normally distributed with evident skewing to the right (Shapiro-Wilk, $p<0.05$) even after log-, square-root and cube-root transformation. Comparison of non-Aroclor PCB concentrations between age groups and between the two locations were analyzed using Wilcoxon Signed Rank Test and Mann-Whitney Rank Sum Test. Spearman correlation coefficient were computed to test for association of PCB 11, 14, 35 and 209 serum concentrations in adolescents with the concentrations in their mothers. P values below 0.05 were considered significant.

RESULTS

In this study, we defined a non-Aroclor PCB congener as any PCB congener that was not commercially produced and detected in USA production of Aroclor 1016, 1221, 1242, 1248, 1254, 1260 and 1262 at a limit of 0.20 weight percent or higher. Due to variability in the manufacture of Aroclors, some of these non-Aroclor congeners may have been present in small quantities in commercial mixtures. We measured PCBs in Aroclors using the same methods used for our samples and we compared our findings to those reported by Frame *et al*[29] and Nakano *et al* on PCBs in USA production.[30] We found no statistically significant difference between average weight percent of each congener detected in our laboratory and Frame *et al*[29] (p=0.986). A total of 70 PCB congeners represented in 68 chromatographic peaks were identified as non-Aroclor PCB congeners (Table 2). Weight percent values for each congener in Aroclor mixtures measured by our laboratory appear in SI, Table S3. The definition of a non-Aroclor we are using here is based entirely on reported and measured congeners in Aroclor mixtures and we do not suggest that we know the source of the PCB congeners found in human serum. We have no way to distinguish if PCBs measured in

human serum were from Aroclor sources or from non-Aroclor sources such as paint pigments. For these PCB congeners, we are not able to distinguish the source.

The concentration, median and sum non-Aroclor PCBs are reported in ng/g fresh weight (f.w.). Among the 70 non-Aroclor PCB congeners, 68 of them were detected in our samples with 11 of them found in at least 25% of our participants (Figure 1). PCB 209 (77%), PCB 14 (73%), PCB 133 (70%), PCB 35 (61%) and PCB 11 (40%) were the five most frequently detected congeners (Figure 1). PCB 11, PCB 14, PCB 35 and PCB 209 were also the major dominating congeners (Figure 2). The sum of non-Aroclor PCBs in participant sera ranged from none detected to 0.288 ng/g f.w. with a median of 0.019 ng/g f.w. Adolescents had significantly lower Σ_{70} non-Aroclor PCBs than mothers in East Chicago ($p<0.001$) and Columbus Junction ($p=0.008$). Columbus Junction participants had significantly higher concentrations of total non-Aroclor PCBs ($p<0.001$). On average, non-Aroclor PCBs constituted about 10% of the Σ_{209} PCBs in our participants, although in some participants, the non-Aroclors constituted as much as 50% the total PCBs detected (Figure 3a). Among these concentrations in our participants, an average of 50% (up to 100%) are likely due to paint exposure (Figure 3b).

PCB 11 (3, 3′-dichlorobiphenyls), was detected in 40% of the participants and usually was the highest in concentration. The concentration ranged from none detected to 0.215 ng/g f.w. Columbus Junction participants had significantly higher levels of PCB 11 than East Chicago participants ($p<0.001$) and there was no significant difference between age groups in East Chicago ($p=0.057$) and Columbus Junction ($p=0.313$) (Figure 1). PCB 11 serum concentration in adolescents and their mothers were strongly correlated (East Chicago, $\rho = 0.62$, $p<0.0001$); Columbus Junction, $\rho = 0.69$, $p<0.0001$) (Table 3).

PCB 14 (3, 5-dichlorobiphenyls) was detected in 73% of the participants ranging from none detected to 0.004 ng/g f.w. and a median of 0.002 ng/g f.w. We found significant difference in PCB 14 concentration between adolescents and their mothers from East Chicago ($p=0.009$) but not Columbus Junction ($p=0.710$) and Columbus Junction communities had significantly higher levels of PCB 14 ($p<0.001$). There was strong correlation of serum PCB 14 in adolescents with that in their mothers in East Chicago ($\rho = 0.59$, $p<0.0001$) and Columbus Junction ($\rho = 0.61$, $p<0.0001$) (Table 3).

One of the non-*ortho* PCB congeners, PCB 35 (3, 3′, 4,-trichlorobiphenyls), was detected in 107 participants. The concentration ranged from none detected to 0.007 ng/g f.w. (median = 0.001 ng/g f.w.). The level of PCB 35 in adolescents was not significantly different from their mothers (East Chicago, $p=0.659$; Columbus Junction, $p=0.382$). We also observed that the community in Columbus Junction had significantly higher PCB 35 than the East Chicago community ($p<0.001$). In East Chicago, no significant association was found for PCB 35 in adolescents with that in their mothers ($\rho = 0.30$, $p=0.059$). Nonetheless, we found moderate and significant association between adolescents and their mothers in Columbus Junction households ($\rho = 0.44$, $p<0.001$).

Over three-fourths of the participants in this study (77%) had detectable PCB 209 (2, 2′, 3, 3′, 4, 4′, 5, 5′, 6, 6′-decachlorobiphenyls) in their serum ranging from none detected to 0.015

ng/g f.w. (median = 0.001 ng/g f.w.). Adolescents had significantly lower PCB 209 than their mothers in both locations (East Chicago, $p<0.001$; Columbus Junction, $p<0.001$). However, there was no significant difference in PCB 209 concentration between locations ($p=0.115$). Moderate correlation was observed between the age groups in Columbus Junction ($\rho = 0.40$, $p=0.003$) while there was no significant correlation among East Chicago participants ($\rho = 0.21$, $p=0.191$) (Table 3).

DISCUSSION

We defined a list of 70 PCB congeners as non-Aroclor PCB congeners and report the prevalence of these congeners in human serum for the first time. The weight percent of each congener in Aroclor mixtures that were measured and analyzed by our laboratory is comparable to those reported by Frame *et al.*[29] This demonstrated that our analytical methods were able to capture and quantify all the PCB congeners in these mixtures accurately compared to Frame and coworkers[29] even though different analytical instruments and methods were used. There were more than 70 congeners having ≤0.20 weight percent in Aroclor; however, we were not able to separate all congeners individually. Thus, we eliminated those congeners that co-eluted with Aroclor congeners. PCB 209, PCB 14, PCB 133, PCB 35 and PCB 11 were the most frequently detected non-Aroclor congeners in our participants, in that order. We are not aware of any previous reports of PCB 14, PCB 35 and PCB 133 in environment matrices. These congeners were first reported in human serum previously by our group[13, 14]. These congeners are generally excluded from commonly performed analytical methods, including EPA methods 8082[31], 608[32], and the EPAs IADN[33] and LMMB[34] methods. On average, about 10% of the total PCBs in our participants came from non-Aroclor PCBs which suggests that serum PCB levels may be underestimated if only accounting for Aroclor PCBs when reporting total PCBs. We further investigated and found that an average 50% and up to 100% of these non-Aroclor PCB concentrations in our participants are likely due to paint exposure.

PCB 11 has been reported in air[16, 17, 35–41], water[18, 19], wastewater treatment plant[20, 42, 43], sediment[10, 19, 20, 24] and biota[19, 20] globally. We have previously reported that PCBs are by-products in US commercial yellow paint pigments.[21] Rodenburg and coworkers have reported their presence in consumer goods that use these pigments[43, 44] to which humans can be exposed. To our knowledge, no study has ever measured PCB 11 in food products although a study found it in animal feeds in Spain.[45] Our laboratory was the first to report PCB 11 in human serum[13, 14] and we know no other reports of PCB 11 in humans. Our group has performed the only inhalation studies of PCB 11[46] and ^{14}C-labelled PCB 11.[47] Our current study found that PCB 11 concentration was not age-dependent which suggests this compound does not biomagnify and its presence may be due to recent exposure. Regardless of location, PCB 11 concentration in adolescents was significantly and strongly associated with their mothers, suggesting that the family environment or diet may be the source of PCB 11. We reported high concentration of PCB 11 in indoor air of our participants' homes and schools in East Chicago and Columbus Junction.[17] As part of that study, we calculated PCB 11 inhalation rates and found adolescents had greater inhalation exposure than mothers due to spending times in schools with high total PCB levels in indoor air.[17]

Understanding of PCB 11 toxicity is growing. Our studies have shown that PCB 11 metabolizes rapidly[46, 47] and its hydroxylated metabolite (4 OH-PCB 11) was found to induce cytoxicity *in vitro*.[48] PCB 11 rapidly metabolized into Phase II metabolites such as PCB 11 sulfate from its hydroxylated metabolite.[47] The lower-chlorinated PCB sulfate congeners, including PCB 11 sulfate, are high-affinity ligands to the human thyroid hormone transport protein transthyretin and may act as thyroid hormone disruptors.[49] All these suggest that PCB 11 is a potential health hazard.

PCB 14 was one of the most frequently detected congeners in our cohorts. To our knowledge, it has rarely been measured nor has it been reported in any matrix.[14] Besides, we did not report PCB 14 in paint pigment because it was used as surrogate standard when we measured PCB congener in paint pigments.[21] Regardless of location, PCB 14 concentrations and detection frequency was the same across age groups. Similar to our finding for PCB 11, there was a significantly strong positive correlation between adolescents and their mothers. We do not know if PCB 14 has any important inhalation sources because it has not been reported in air. Also, similar to our finding for PCB 11, the Columbus Junction community had significantly higher concentration of PCB 14 than East Chicago community. This suggests that PCB 14 is omnipresent in both urban and rural locations. To our knowledge, no published information is available about the toxicity of PCB 14 as the presence of this congener in humans and the environment has been overlooked.

PCB 35 was found in more than half of the participants in our study. This non-*ortho* PCB congener could be a dechlorination product of PCB 77. PCB 77 is a dioxin-like compound and we have reported it in Chicago air[8] and in commercial paint pigments.[21, 22] PCB 35 was also detected in aryl azo pigments in commercially available paints in Japan.[22] Anezaki *et al* suggested that PCB 35 was generated as byproduct with the process similar to the formation of PCB 11 when aryl azo paint pigments were manufactured.[22] This could possibly be one of the sources of human exposure to PCB 35. We found that the concentration in human serum was not age-dependent and hence there was likely limited biomagnification over time. This lower-chlorinated congener is suspected to metabolize faster than higher-chlorinated PCBs. No significant association of PCB 35 concentration in East Chicago adolescents with their mothers was observed whereas moderate association was found among participant groups from the Columbus Junction community. Columbus Junction participants had significantly higher PCB 35 levels than those from East Chicago. We cannot confirm if paint pigment was the sole source of exposure, and it is unknown if other building materials, personal care products, or food could have produced exposure to PCB 35. No published information is available on PCB 35 toxicity; however, studies have investigated the toxicity of its possible hydroxylated metabolites. 4′-OH-PCB 35 was found to induce reactive oxygen species *in vitro*.[50] Using quantitative structure-activity relationship techniques, PCB 35 and 6′-OH-PCB 35 were predicted to have high probability of carcinogenicity in female mice and PCB 35 was also found to be mutagenic.[51]

PCB 209 has been reported in food products including freshwater fish.[25, 26] Duarte-Davidson *et al* estimated that total daily intake of PCB 209 in United Kingdom populations was about 2.1 nanograms.[52] Very high concentrations of PCB 209 were measured in phthalocyanine-type pigments, especially green pigment, used in commercially available

paint.[21, 22] It has also been measured in liver and fat of three types of sea turtles in the Canary Islands, Spain.[53] A few studies detected PCB 209 in human blood and serum.[14, 15, 27, 54] Our current study found 134 participants (about 77%) contained PCB 209 in their serum (79 adults and 55 children). We found no significant difference in its level between the communities. This suggests that human exposure to PCB 209 could be due to contact with items containing phthalocyanine-type pigments and/or consuming contaminated food products. Adolescents had significantly lower PCB 209 concentration than mothers and it was poorly correlated between adolescents and their mothers which suggests greater bioaccumulation of PCB 209 in mothers.

We measured non-Aroclor PCB congeners in human serum from adolescents and their mothers living in two United States Midwestern communities and report their prevalence. These congeners represented an average of 10% (and up to 50%) of total PCBs measured in human serum with PCB 11, 14, 35 and 209 as the major congeners. An average of 50% of these non-Aroclor PCB concentrations were attributed to aryl azo and phthalocyanine paint pigments. PCB 11 and PCB 209 were measured in organic paint pigments, consumer goods and some food products. Very little is known about other non-Aroclor PCB congeners including their origin, their fate in the environment, and their absorption, distribution, metabolism, excretion and toxicity in humans.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

Acknowledgments

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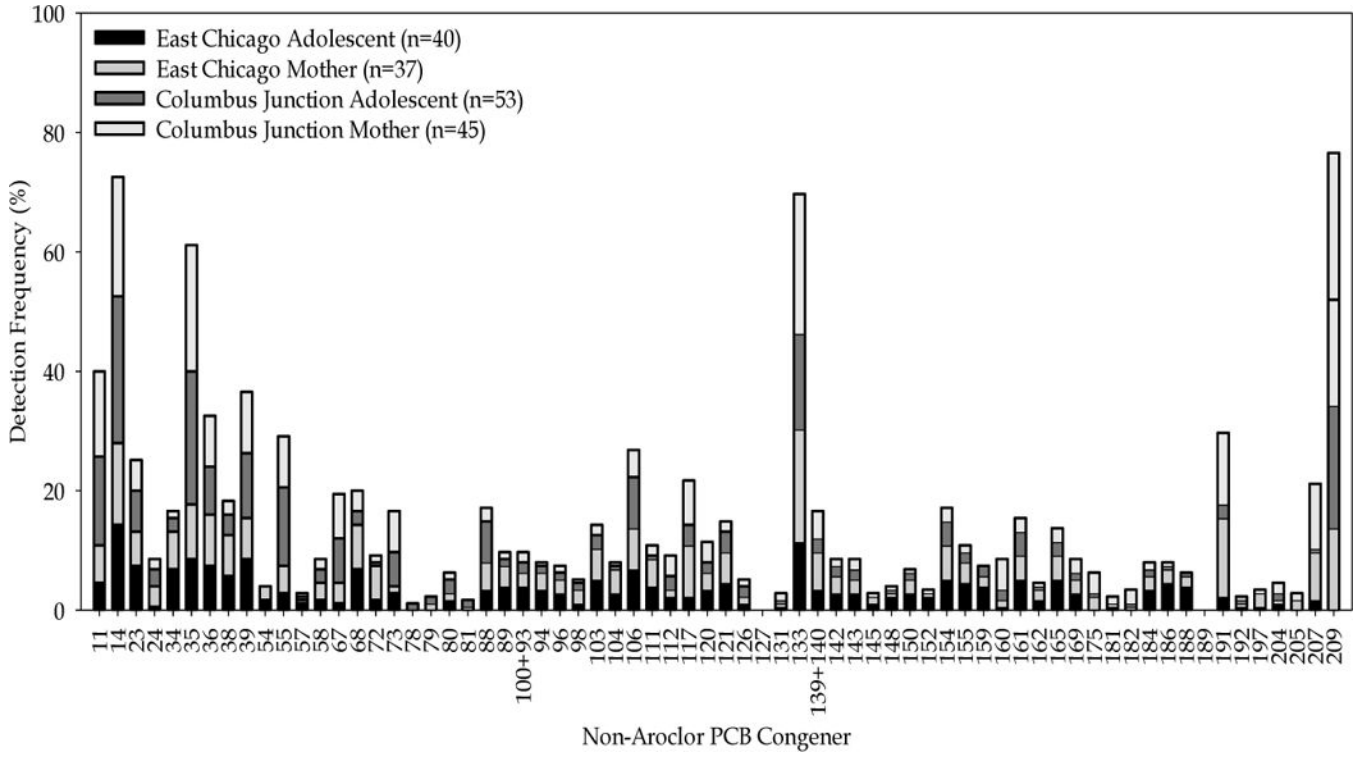

Figure 1.

Detection frequency of each non-Aroclor PCB congener in adolescents and their mothers in East Chicago and Columbus Junction. (Bars indicate proportion of the total 175 participants)

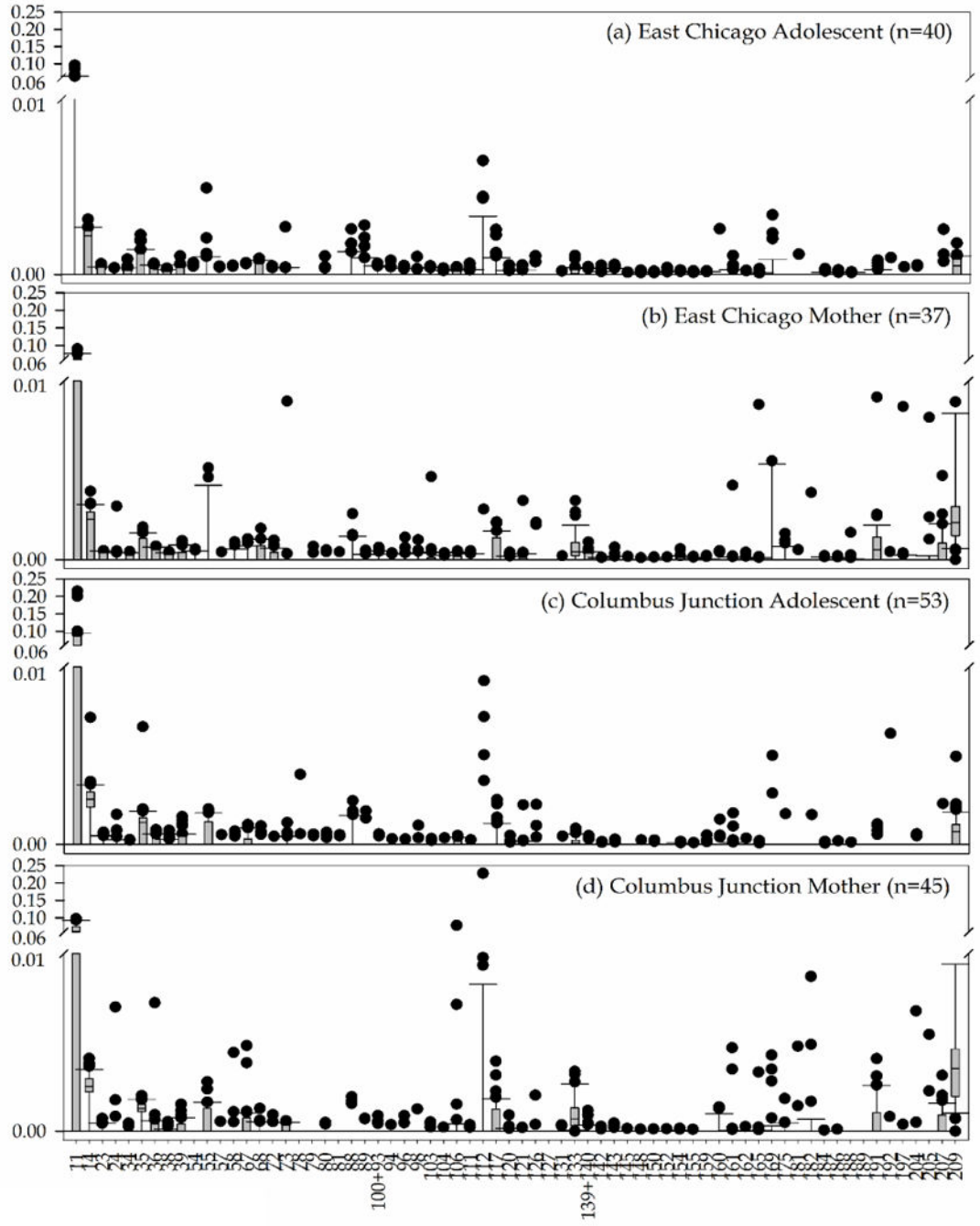

Figure 2.

Non-Aroclor PCB congener profile of adolescents and their mothers in East Chicago and Columbus Junction.

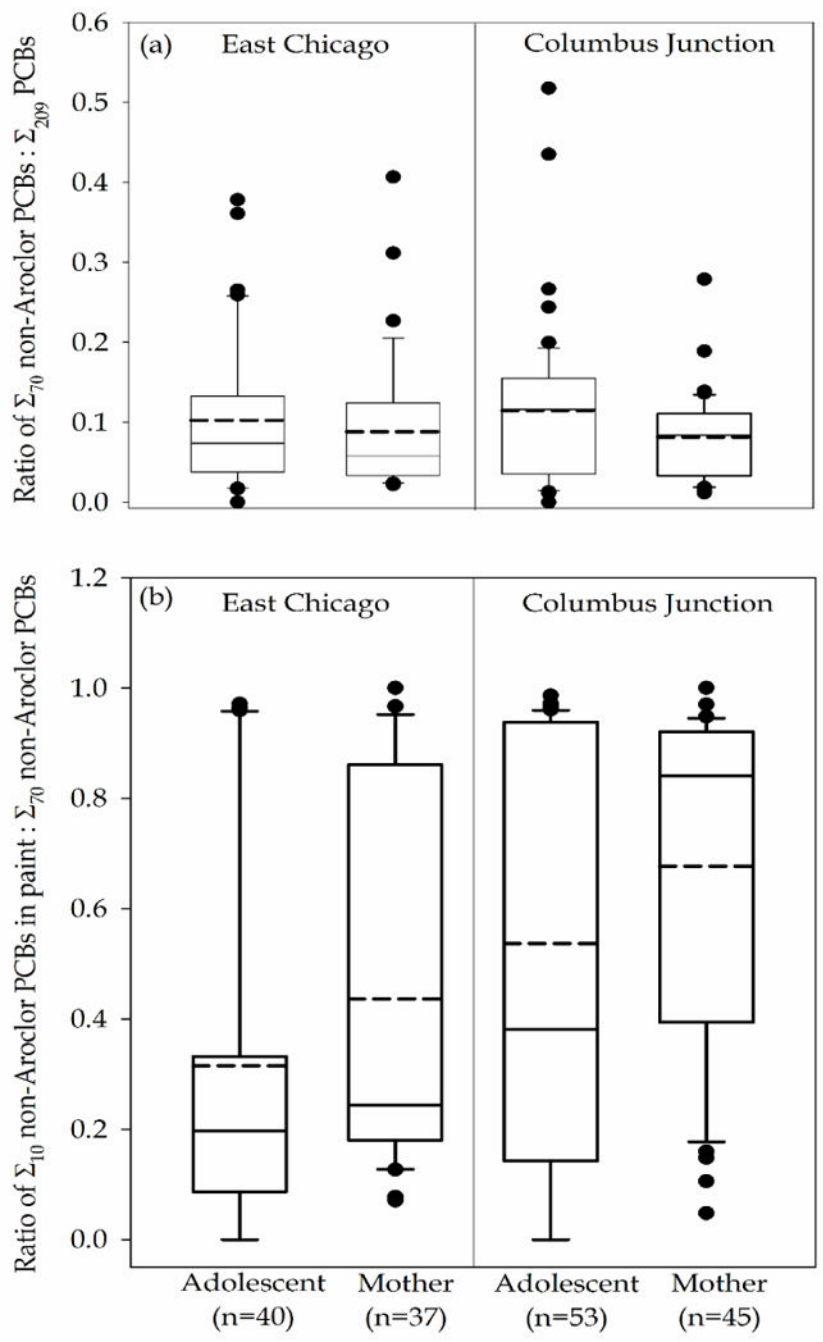

Figure 3.

Ratio of sum 70 non-Aroclor PCB congeners to sum 209 PCB congeners (a) and sum 10 non-Aroclor PCB congeners in paint to sum 70 non-Aroclor PCB congeners (b) in adolescents and their mothers in East Chicago and Columbus Junction. (Dash line represents mean line)

Table 1

Demographic data of East Chicago and Columbus Junction

	East Chicago	Columbus Junction
Adolescent		
Sample size	40	53
Age, years	13–18	14–17
Gender		
Female	20	27
Male	20	26
Race/Ethnicity		
Hispanic	30*	34
White (non-Hispanic)	2	17
African American	12	1
Other	0	1
Mother		
Sample size	37	45
Age, years	29–58	34–53
Race/Ethnicity		
Hispanic	28*	25
White (non-Hispanic)	2	19
African American	8	0
Other	0	1

* 4 adolescents and 1 mother self-identify as Arfican-American Hispanic

Table 2

A list of 70 non-Aroclor PCB congeners defined as PCB congeners detected in ≤0.20% in Aroclor 1016, 1221, 1242, 1248, 1254, 1260 or 1262. (Congeners in bold and underline are congeners that are also detected in paint pigments. Paint pigment type and color are listed at the table bottom)

Non-Aroclor PCB Congener			
11	**78**	121	162
14	79	126	165
23	80	127	169
24	81	131	175
34	88	133	181
35	89	139+140	182
36	100+93	**142**	184
38	94	143	186
39	96	145	188
54	98	148	189
55	103	150	191
57	104	152	192
58	**106**	154	197
67	111	155	204
68	112	159	**205**
72	117	**160**	**207**
73	120	**161**	**209**

PCB 11: azo-type paint pigments (yellow, green and red)[21, 22]

PCB 35: azo-type paint pigments[22]

PCB 78: medium yellow[21]

PCB 106, 142: green[21]

PCB 160: blue and green[21]

PCB 161: magenta[21]

PCB 205: phthalocyanine-type paint pigments[22]

PCB 207: phthalocyanine-type paint pigments (green)[21, 22]

PCB 209: phthalocyanine-type paint pigments (green, carbazole violet, blue)[21, 22]

Table 3

Test of association using Spearman correlation for adolescent-mother pairs in East Chicago and Columbus Junction

	Spearman correlation, ρ	
Congener	**East Chicago**	**Columbus Junction**
PCB 11	0.62**	0.69**
PCB 14	0.59**	0.61**
PCB 35	0.30	0.44*
PCB 209	0.21	0.40*

* $p < 0.05$ and

** $p < 0.001$